	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	58	pr-39	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:44			0
2	BRS	L3	1087	proteasome	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:44			0
3	BRS	L2	3	pr-39 same oligopeptide	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:46			0
4	BRS	L4	2	1 same 3	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:46			0
5	BRS	L5	558	proteasome same (inhibit$3)	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:52			0
6	BRS	L6	2	1 same 5	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:53			0
7	BRS	L8	0	gao adj youhe.in.	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:54			0
8	BRS	L9	0	7 and 1	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:55			0
9	BRS	L7	194	simons adj michael.in.	USPAT; US-PGPUB; EPO; JPO;	2003/07/20 15:56			0

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FILE 'MEDLINE' ENTERED AT 16:02 ON 20 JUL 2003

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FILE 'SCISEARCH' ENTERED AT 16:02:13 ON 20 JUL 2003
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FILE 'AGRICOLA' ENTERED AT 16:02:13 ON 20 JUL 2003

=> s Pr-39
L1          425 PR-39

=> s proteosome
L2         1372 PROTEOSOME

=> s l2 (p) inhibit?
L3          462 L2 (P) INHIBIT?

=> s l1 (p) l3
L4            0 L1 (P) L3

=> s simons michael/au
L5          303 SIMONS MICHAEL/AU

=> s gao youhe/au
L6           25 GAO YOUHE/AU

=> s (l5 or l6) and l1
L7           13 (L5 OR L6) AND L1

=> duplicate remove l7
DUPLICATE PREFERENCE IS 'CAPLUS, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L7
L8           11 DUPLICATE REMOVE L7 (2 DUPLICATES REMOVED)

=> d l8 1-11 ibib abs

L8   ANSWER 1 OF 11  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER:    2003:80682  BIOSIS
DOCUMENT NUMBER:     PREV200300080682
TITLE:               Adenoviral   ***PR***  -  ***39***   improves perfusion and
                     function in a pig model of chronic myocardial ischemia.
AUTHOR(S):           Post, Mark J. (1); Bao, Jialin; Sato, Kaori; Murakami,
                     Masahiro; Pearlman, Justin D.;   ***Simons, Michael***
CORPORATE SOURCE:    (1) Dartmouth Medical Sch, Lebanon, NH, USA USA
SOURCE:              Circulation, (November 5 2002) Vol. 106, No. 19 Supplement,
                     pp. II-275. print.
                     Meeting Info.: Abstracts from Scientific Sessions Chicago,
                     IL, USA November 17-20, 2002 American Heart Association
                     . ISSN: 0009-7322.
DOCUMENT TYPE:       Conference
LANGUAGE:            English

L8   ANSWER 2 OF 11  CAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2001:489246  CAPLUS
DOCUMENT NUMBER:          135:87168
TITLE:                    Method for   ***PR***  -  ***39***   peptide-mediated
                          selective inhibition of I.kappa.B.alpha. degradation
INVENTOR(S):                ***Simons, Michael***  ;   ***Gao, Youhe***
PATENT ASSIGNEE(S):       Beth Israel Deaconess Medical Center, USA
SOURCE:                   PCT Int. Appl., 68 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:
```

PATENT NO.	KIND	DAT[redacted]	APPLICATION NO.	DAT[redacted]
WO 2001047540	A1	20010705	WO 2000-US35293	20001227
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
EP 1242107	A1	20020925	EP 2000-989492	20001227
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				

PRIORITY APPLN. INFO.: US 1999-474967 A 19991229
WO 2000-US35293 W 20001227

AB The invention provides both a method and means for regulating I.kappa.B.alpha. degrdn., NF.kappa.B activity, and NF.kappa.B-dependent gene expression within living cells, tissues, and organs in-situ. The selective regulation is performed using native ***PR*** - ***39*** peptide or one of its shorter-length homologs, for interaction with such I.kappa.B.alpha. and proteasomes as are present in the cytoplasm of viable cells. The result of ***PR*** - ***39*** peptide interaction with I.kappa.B.alpha. is a selective alteration in the intracellular proteolytic activity of proteasomes, which in turn, causes a redn. of I.kappa.B.alpha., a decrease of NF.kappa.B activity, and a down-regulation of NF.kappa.B-dependent gene expression.

L8 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:319740 CAPLUS
DOCUMENT NUMBER: 134:336214
TITLE: Method for ***PR*** - ***39*** peptide regulated stimulation of angiogenesis
INVENTOR(S): ***Simons, Michael*** ; ***Gao, Youhe***
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001030368	A1	20010503	WO 2000-US27552	20001006
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				

PRIORITY APPLN. INFO.: US 1999-426011 A 19991025

AB The present invention provides both a method and means for regulating angiogenesis within living cells, tissues, and organs in-situ. The regulation is performed using native ***PR*** - ***39*** peptide or one of its shorter-length homolog, for interaction with such proteasomes as one present in the cytoplasm of viable cells. The result of ***PR*** - ***39*** peptide interaction with proteasomes is a decrease in the intracellular degrdn. of active peptides such as HIF-1.alpha. and a consequential stimulation of angiogenesis in-situ.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2001:936421 CAPLUS
DOCUMENT NUMBER: 136:178301
TITLE: ***PR*** - ***39*** and PR-11 peptides inhibit ischemia-reperfusion injury by blocking proteasome-mediated I.kappa.B.alpha. degradation
AUTHOR(S): Bao, Jialin; Sato, Kaori; Li, Min; ***Gao, Youhe*** ; Abid, Ruhul; Aird, William; ***Simons, Michael*** ; Post, Mark J.
CORPORATE SOURCE: Angiogenesis Research Center, Beth Israel Deaconess Medical Center, Dartmouth Medical School, Hanover, NH, 03756, USA
SOURCE: American Journal of Physiology (2001), 281(6, Pt. 2), H2612-H2618
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB ***PR*** - ***39*** inhibits proteasome-mediated I.kappa.B.alpha. degrdn. and might protect against ischemia-reperfusion injury. The authors studied ***PR*** - ***39*** , its truncated form PR-11, and a

mutant PR-11AAA, which lacks the ability to prevent I.kappa.B.alpha. degrdn., in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg ***PR*** - ***39*** or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, resp., which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion mol.-1 and vascular cell adhesion mol. 1 were reduced. Thus, ***PR*** - ***39*** and PR-11 effectively inhibit myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by inhibition of I.kappa.B.alpha. degrdn. and subsequent inhibition of nuclear factor-.kappa.B-dependent adhesion mols. The active sequence is located in the first 11 amino acids, suggesting a potential for oligopeptide therapy as an adjunct to revascularization.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 5 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:275090 BIOSIS
DOCUMENT NUMBER: PREV200200275090
TITLE: Inhibition of apoptosis by ***PR*** - ***39*** is mediated with increased IAP2 expression.
AUTHOR(S): Li, Jian (1); Post, Mark J. (1); ***Simons, Michael*** ***
(1)***
CORPORATE SOURCE: (1) Beth Israel Deaconess Med Ctr, Harvard Med Sch, Boston, MA USA
SOURCE: Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.293-II.294. http://circ.ahajournals.org/. print. Meeting Info.: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L8 ANSWER 6 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:263290 BIOSIS
DOCUMENT NUMBER: PREV200200263290
TITLE: ***PR*** - ***39*** and PR-11 peptides protect against ischemia-reperfusion injury by inhibition of proteasome mediated IkappaBalpha degradation.
AUTHOR(S): Bao, Jialin (1); ***Gao, Youhe (1)*** ; Li, Min (1); Abid, Md. Ruhul (1); Aird, William (1); ***Simons,*** ***
Michael (1)*** ; Post, Mark Johannes (1)
CORPORATE SOURCE: (1) Beth Israel Deaconess Med Ctr, Harvard Med Sch, Boston, MA USA
SOURCE: Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.52. http://circ.ahajournals.org/. print. Meeting Info.: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L8 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:706997 CAPLUS
DOCUMENT NUMBER: 133:276343
TITLE: Method for ***PR*** - ***39*** peptide regulated stimulation of angiogenesis
INVENTOR(S): ***Simons, Michael*** ; ***Gao, Youhe***
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000057895	A1	20001005	WO 2000-US7050	20000316

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,

PT, SE
EP 1165111 A1 200[redacted]02 EP 2000-919442 200[redacted]6
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
PRIORITY APPLN. INFO.: US 1999-276868 A 19990326
WO 2000-US7050 W 20000316
AB The present invention provides both a method and means for regulating angiogenesis within living cells, tissues, and organs in-situ. The regulation is performed using native ***PR*** - ***39*** peptide or one of its shorter-length homologs, for interaction with such proteasomes as one present in the cytoplasm of viable cells. The result of ***PR*** - ***39*** peptide interaction with proteasomes is a decrease in the intracellular degrdn. of active peptides such as HIF-1.alpha. and a consequential stimulation of angiogenesis in-situ.
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:178321 CAPLUS
DOCUMENT NUMBER: 133:205925
TITLE: PR39, a peptide regulator of angiogenesis. [Erratum to document cited in CA132:149677]
AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; ***Gao,*** Youhe*** ; Li, Min; Metals, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; ***Simons, Michael***
CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SOURCE: Nature Medicine (New York) (2000), 6(3), 356
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The correct versions are given for Figs. 2a, c, and d on page 51; Fig. 3c on page 52; and Fig. 5b on page 53.

L8 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:46162 CAPLUS
DOCUMENT NUMBER: 132:149677
TITLE: PR39, a peptide regulator of angiogenesis
AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; ***Gao,*** Youhe*** ; Li, Min; Metais, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; ***Simons, Michael***
CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery both at Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SOURCE: Nature Medicine (New York) (2000), 6(1), 49-55
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Although tissue injury and inflammation are considered essential for the induction of angiogenesis, the mol. controls of this cascade are mostly unknown. Here we show that a macrophage-derived peptide, PR39, inhibited the ubiquitin-proteasome-dependent degrdn. of hypoxia-inducible factor-1.alpha. protein, resulting in accelerated formation of vascular structures in vitro and increased myocardial vasculature in mice. For the latter, coronary flow studies demonstrated that PR39-induced angiogenesis resulted in the prodn. of functional blood vessels. These findings show that PR39 and related compds. can be used as potent inductors of angiogenesis, and that selective inhibition of hypoxia-inducible factor-1.alpha. degrdn. may underlie the mechanism of inflammation-induced angiogenesis.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 10 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:524759 BIOSIS
DOCUMENT NUMBER: PREV199900524759
TITLE: Cardiac-specific overexpression of ***PR*** - ***39*** induces angiogenesis, myocardial hypertrophy, and increased microvascular reactivity.
AUTHOR(S): Li, Jian; Hampton, Thomas G.; Metais, Caroline; Ma, Lijie;

Li, Jianyi; Amende, Ivo; Sellke, Frank W.; Douglas, Pamela S.; Morgan, James P.; ***Simons, Michael***
CORPORATE SOURCE: BIBMC/Harvard Med. Sch., Boston, MA USA
SOURCE: Circulation, (Oct. 27, 1998) Vol. 98, No. 17 SUPPL., pp. I794.
Meeting Info.: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998
The American Heart Association
. ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L8 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2003 ACS on STNDUPLICATE 2
ACCESSION NUMBER: 1997:708269 CAPLUS
DOCUMENT NUMBER: 128:2511
TITLE: Macrophage-dependent regulation of syndecan gene expression
AUTHOR(S): Li, Jian; Brown, Lawrence F.; Laham, Roger J.; Volk, Rudiger; ***Simons, Michael***
CORPORATE SOURCE: Angiogenesis Research Center, Cardiovascular Division, Department of Medicine, Harvard Medical School, Boston, MA, USA
SOURCE: Circulation Research (1997), 81(5), 785-796
CODEN: CIRUAL; ISSN: 0009-7330
PUBLISHER: American Heart Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Heparan sulfates in the extracellular matrix are required for a variety of biol. processes, including cellular response to heparin-binding growth factors. However, little is known regarding the regulation of their expression and compn. under pathophysiol. conditions. In the present study, the authors have investigated the regulation of expression of two key heparan sulfate chain-carrying core proteins, syndecan-1 and syndecan-4, in a mouse/rat infarct model of tissue injury and repair. Induction of myocardial infarction was assocd. with a prompt increase in expression of both syndecan genes. Although infiltrating macrophages accounted for a substantial increase in syndecan expression, increased expression was noted in the levels of syndecan-1 mRNA in endothelial cells and syndecan-4 mRNA in cardiac myocytes. This increase in expression was limited to the immediate peri-infarct region and was absent from remote areas of the left or right ventricles. The influx of blood-derived macrophages in the heart correlated with the appearance of ***PR*** - ***39*** peptide, which has previously been shown to increase syndecan expression in vitro. Studies in the op/op mice strain (which demonstrates sharply reduced levels of circulating monocytes) showed that myocardial infarction was assocd. with markedly reduced levels of macrophage influx and corresponding redn. in the expression of ***PR*** - ***39*** and both syndecan genes. Pretreatment of op/op mice with granulocyte macrophage colony-stimulating factor restored myocardial macrophage content with corresponding restoration of ***PR*** - ***39*** /syndecan expression. In summary, myocardial infarction is assocd. with a distinct spatial and temporal pattern of syndecan-1 and -4 gene expression, which is induced by an influx of blood-derived macrophages.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 16:02:13 ON 20 JUL 2003
L1 425 S PR-39
L2 1372 S PROTEOSOME
L3 462 S L2 (P) INHIBIT?
L4 0 S L1 (P) L3
L5 303 S SIMONS MICHAEL/AU
L6 25 S GAO YOUHE/AU
L7 13 S (L5 OR L6) AND L1
L8 11 DUPLICATE REMOVE L7 (2 DUPLICATES REMOVED)

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